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Commentary

Concluding Remarks on Session II

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The assessment of human cancer risk from exposure to chemicals in aquatic environments includes understanding such factors as chemical inputs, bioaccumulation, metabolism, food-chain transfer and biological effects. The estimation of the risk to humans is dependent on being able to evaluate the net effect of these complementary parameters on human populations, recognizing that humans are ultimately affected by chemical changes that have occurred in the aquatic environment.

The task we face is obviously profoundly difficult. It is particularly so because almost all aquatic environments are comprised of complex mixtures of chemicals that can act synergistically or antagonistically to produce a given effect. These effects can range from subtle biochemical changes to alterations in behavior. However, exactly how these events relate to cancer, *per se*, is shrouded in mystery.

The difficult task of human risk assessment is exacerbated by large gaps in knowledge embracing virtually every area necessary for the attainment of valid judgments. Chemical inputs are only partially understood, as is the actual chemical composition of sediments and tissues. While we often operate on the assumption that these matrices from urban areas are composed of simple suites of chemicals, such as certain metals, PCBs, and aromatic hydrocarbons, we know that this is not actually true. Scores of other contaminants cannot simply be dismissed as influencing toxicity just because we do not or cannot analyze for them.

Neither can we assume (as we sometimes do) that an individual compound that fascinates us scientifically, or otherwise catches our attention, is necessarily singularly important in the manifestation of toxic effects. Benzo(a)pyrene, for example, is an interesting model. It has been studied extensively, but are BaP and its metabolites especially important in assessing toxicity in the presence of scores of other environmentally derived compounds in tissues? One suspects not, but there is little information upon which to base an argument either way.

Further, how reasonable is it to dismiss the contributions of agents, such as free radicals arising from environmental chemicals, in the induction of cancer? Such compounds may be necrogenic, for example, and thus play a major role in cell proliferation. Free radicals are only one example in this context scores of other chemicals present in sediments and tissues of aquatic species are potential candidates for influencing (e.g., promoting) the effects of carcinogenic chemicals.

So where do we stand in our attempts to relate the contamination of aquatic life to human cancer? I submit that we find ourselves in a weak, unsatisfactory position. We know something about the composition of sediments and considerably less about the accumulation of chemicals in edible tissues of fish and shellfish. Our understanding of metabolism in aquatic systems is sketchy and based on studies of just a few compounds. Moreover, much of this work is hard to relate to effects on human populations.

We know that certain compounds that accumulate in tissues of fish and shellfish are carcinogenic, and we then can calculate risk factors. Yet, these values are suspect for a number of reasons, one of which is the fact that they are based on the effects of only one or two compounds (usually PCBs or DDT derivatives) and synergism/antagonism is discounted. Specifically, in relation to human impacts, we have only a marginal understanding of populations at risk, as well as of differential effects on infants and pregnant women. Many other issues are equally elusive, yet eminently worthy of attention.

Some argue that risk assessments, while fraught with problems, nevertheless provide a wide margin of safety for the public. This may be so in some cases, but commercial fishing interests become understandably alarmed by the high human cancer risks projected for the consumption of fish from urban areas ranging from California to Massachusetts. There seems to be no excuse for accepting flawed assessment techniques when we can do better—perhaps not right away, but progressively through long-term, dedicated research to fill the gaps and thus reduce the uncertainty of our assessments. The type of research described in the present session should become an important, integral part of our future effort.

The presentations at this session demonstrated a number of deficiencies in our knowledge. However, and most significantly, they also showed that credible research can and does lead to a progressive increase in understanding. However, the problem is that progress has been very slow due to a lack of funds and a lack of dedication to important areas of research and other factors. These problems can be rectified. The solutions are difficult to attain, but they are tractable. The fact is that these problems must be rectified if any real understanding is to be obtained on the effects of aquatic pollution on human cancer. Further discussion at this juncture is almost nonproductive. What we need are credible facts if we are going to put the puzzle together—for this there is no substitute.

Perhaps the basic question is not how we will solve the problem, but when will we start to address it in a truly productive way? Right now, our only hope seems to be that random pieces

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of information, gathered by various scientists for different reasons, will somehow come together to provide the necessary light in the darkness. One might argue that there is not even the semblance of an integrated effort, no master plan to guide our efforts. Yet one hopes that the present conference has provided a catalyst for answering the important question "what is the effect of contaminated fish and shellfish on present and future human populations, particularly with respect to increased cancer risks?" Overall, I believe we have achieved a significant beginning.